Case History & Preoperative Assessment

Surgical procedures and administration of anesthesia are associated with a complex stress response that is proportional to the magnitude of injury, total operating time, amount of intraoperative blood loss and degree of postoperative pain. The adverse metabolic and hemodynamic effects of this stress response can present many problems in the perioperative period. Decreasing the stress response to surgery and trauma is the key factor in improving outcome and lowering the length of hospital stay as well as the total costs of patients care.

It is well recognized that safe and efficient surgical and anesthesia practice requires an *optimized* patient. Several of the large-scale epidemiological studies have indicated that inadequate preoperative preparation of the patient may be a major contributory factor to the primary causes of perioperative mortality.

The following primary goals of preoperative evaluation and preparation have been identified:

1. Documentation of the condition(s) for which surgery is needed.

2. Assessment of the patient's overall health status.

3. Uncovering of hidden conditions that could cause problems both during and after surgery.

4. Perioperative risk determination.

5. Optimization of the patient's medical condition in order to reduce the patient's surgical and anesthetic perioperative morbidity or mortality.

6. Development of an appropriate perioperative care plan.

7. Education of the patient about surgery, anesthesia, intraoperative care and postoperative pain treatments in the hope of reducing anxiety and facilitating recovery.

8. Reduction of costs, shortening of hospital stay, reduction of cancellations and increase of patient satisfaction**.**

The ultimate goals of preoperative medical assessment are to reduce the patient's surgical and anesthetic perioperative morbidity or mortality, and to return him to desirable functioning as quickly as possible. It is imperative to realize that "perioperative" risk is multifactorial and a function of the preoperative medical condition of the patient, the invasiveness of the surgical procedure and the type of anesthetic administered. A history and physical examination, focusing on risk factors for cardiac and pulmonary complications and a determination of the patient's functional capacity, are essential to any preoperative evaluation. Laboratory investigations should be ordered only when indicated by the patient's medical status, drug therapy, or the nature of the proposed procedure and not on a routine basis. Persons without concomitant medical problems may need little more than a quick medical review. Those with comorbidity should be optimized for the procedure. Proper consultations with appropriate medical services should be obtained to improve the patient's health. These consultations should ideally not be done in a "last second" fashion. The preoperative preparation involves procedures that are implemented based on the nature of the expected operation as well as the findings of the diagnostic workup and the preoperative evaluation**.**

General Health Assessment

The history

The history is the most important component of the preoperative evaluation. The history should include a past and current medical history, a surgical history, a family history, a social history (use of tobacco, alcohol and illegal drugs), a history of allergies, current and recent drug therapy, unusual reactions or responses to drugs and any problems or complications ssociated with previous anesthetics.

A family history of adverse reactions associated with anesthesia should also be obtained. In children, the history should also include birth history, focusing on risk factors such as prematurity at birth, perinatal complications and congenital chromosomal or anatomic malformations and history of recent infections, particularly upper and lower respiratory tract infections.

The history should include a complete review of systems to look for undiagnosed disease or inadequately controlled chronic disease. Diseases of the cardiovascular and respiratory systems are the most relevant in respect of fitness for anesthesia and surgery.

Physical examination

The physical examination should build on the information gathered during the history. At a minimum, a focused preanesthesia physical examination includes an assessment of the airway, lungs and heart, with documentation of vital signs. Unexpected abnormal findings on the physical examination should be investigated before elective surgery.

Laboratory work up

It is generally accepted that the clinical history and physical examination represent the best method of screening for the presence of disease. Routine laboratory tests in patients who are essential.

A clinician should consider the risk-benefit ratio of any ordered lab test. When studying a healthy population, 5% of patients will have results which fall outside the normal range. Lab tests should be ordered based on information obtained from the history and physical exam, the age of the patient and the complexity of the surgical procedure.

Indications for specific preoperative tests

Complete blood count Major surgery Chronic cardiovascular, pulmonary, renal, or hepatic disease; or malignancy Known or suspected anemia, bleeding diathesis, or myelosuppression Less than one year of age
International Normalized Ratio (INR), Activated Partial Thromboplastin Time (aPTT) Anticoagulant therapy Bleeding diathesis Liver disease
Electrolytes & creatinine Hypertension Renal disease Diabetes Pituitary or adrenal disease Digoxin or diuretic therapy, or other drug therapies affecting electrolytes
Fasting glucose Diabetes (should be repeated on day of surgery)
Electrocardiograph Heart disease, hypertension, diabetes Other risk factors for cardiac disease (may include age) Subarachnoid or intracranial hemorrhage, cerebrovascular accident, head trauma
Chest radiograph Cardiac or pulmonary disease Malignancy

Drug history

A history of medication use should be obtained in all patients. Especially, the geriatric population consumes more systemic medications than any other group. Numerous drug interactions and complications arise in this population and special attention should be paid to them.

Generally, administration of most drugs should be continued up to and including the morning of operation, although some adjustment in dosage may be required (e.g. antihypertensives, insulin).

Some drugs should be discontinued preoperatively. The monoamine oxidase inhibitors should be withdrawn 2-3 weeks before surgery because of the risk of interactions with drugs used during anesthesia. The oral

contraceptive pill should be discontinued at least 6 weeks before elective surgery because of the increased risk of venous thrombosis.

Recently, the American Society of Anesthesiologists (ASA) examined the use of herbal supplements and the potentially harmful drug interactions that may occur with continued use of these products preoperatively. All patients are requested to discontinue their herbal supplements at least 2 weeks prior to surgery.

The use of medications that potentiate bleeding needs to be evaluated closely, with a risk-benefit analysis for each drug and with a recommended time frame for discontinuation based on drug clearance and half life characteristics. Aspirin should be discontinued 7-10 days before surgery to avoid excessive bleeding and thienopyridines (such as clopidogrel) for 2 weeks before surgery. Selective cyclooxygenase-2 (COX-2) inhibitors do not potentiate bleeding and may be continued until surgery. Oral anticoagulants should be stopped 4-5 days prior to invasive procedures, allowing INR to reach a level of 1.5 prior to surgery**.**

Perioperative risk assessment

Perioperative risk is a function of the preoperative medical condition of the patient, the invasiveness of the surgical procedure and the type of anesthetic administered. The *ASA grading system* was introduced originally as a simple description of the physical state of a patient. Despite its apparent simplicity, it remains one of the few prospective descriptions of the patient general health which correlates with the risk of anesthesia and surgery. It is extremely useful and should applied to all patients who present for surgery. Increasing physical status is associated with increasing mortality.

American Society of Anesthesiologists' Classification of Physical Status

Status	*Disease State*
ASA class 1	No organic, physiologic, biochemical, or psychiatric disturbance
ASA class 2	Mild to moderate systemic disturbance that may or may not be related to the reason for surgery *Examples*: Heart disease that only slightly limits physical activity, essential hypertension, diabetes mellitus, anemia, extremes of age, morbid obesity, chronic bronchitis
ASA class 3	Severe systemic disturbance that may or may not be related to the reason for surgery, *(does limit activity)* *Examples*: Heart disease that limits activity, poorly controlled essential hypertension, diabetes mellitus with vascular complications, chronic pulmonary disease that limits activity, angina pectoris, history of prior myocardial infarction
ASA class 4	Severe systemic disturbance that is life-threatening with or without surgery *Examples*: Congestive heart failure, persistent angina pectoris, advanced pulmonary, renal, or hepatic dysfunction
ASA class 5	Moribund patient who has little chance of survival but is submitted to surgery as a last resort (resuscitative effort) *Examples*: Uncontrolled hemorrhage as from a ruptured abdominal aneurysm, cerebral trauma, pulmonary embolus.
ASA class 6	A declared brain-dead patient whose organs are being removed for donor purposes
E	An "E" is added to the status number to designate an emergency operation

Surgical complications occur frequently. One large study documented at least one complication in 17% of surgical patients. Surgery-related morbidity and mortality generally fall into one of three categories: cardiac, respiratory and infectious complications. The overall risk for *surgery-related complications* depends on individual factors and the type of surgical procedure. For example, advanced age places a patient at increased risk for surgical morbidity and mortality. The reason for an age-related increase in surgical complications appears to correlate with an increased likelihood of underlying disease states in older persons. Diseases associated with an increased risk for surgical complications include respiratory and cardiac disease, malnutrition and diabetes mellitus. With respect to the type of surgery, major vascular, intraabdominal and intrathroracic surgical procedures, as well as intracranial neurosurgical procedures are frequently associated with increased perioperative morbidity and mortality19-20. In addition, urgent and emergency procedures constitute higher risk situations than elective, nonurgent surgery and present a limited opportunity for preoperative evaluation and treatment.

When one looks at strictly *anesthetic problems* that lead to morbidity and mortality, airway problems and failure to provide adequate ventilation leading to hypoxia become important. Fortunately the number of critical incidents involving anaesthetics alone appear to be decreasing in recent years.

The American College of Cardiology (ACC) and the American Heart Association (AHA) published a task force report on Guidelines for Perioperative Cardiovascular Evaluation for Noncardiac Surgery. The purpose is to provide a framework for considering cardiac risk of noncardiac surgery in a variety of patients and operative situations.

The factors which guide decision making include the patient's cardiovascular risk and functional capacity and the surgery specific risk.

***Patients' risk factors* are usually subdivided into three categories:**

Major-risk factors. A 6-week period is necessary for the myocardium to heal after an infarction and for the thrombosis to resolve. Patients with coronary revascularization done within the preceding 40 days should also be classified as high-risk patients. Because of sympathetic stimulation and hypercoagulability during and after surgery, *patients with major predictors have a five times greater perioperative risk*. Only vital or emergency surgical procedures should therefore be considered for these patients. All elective operations should be postponed and the patients properly investigated and treated**.**

Intermediate-risk factors are proof of well-established but controlled coronary artery disease. Diabetes mellitus is included in this category because it is frequently associated with silent ischemia and represents an independent risk factor for perioperative mortality.

Minor risk factors are markers of an increased probability of coronary artery disease, but not of an increased perioperative risk**.**

The decision to proceed with *elective* surgery begins with an assessment of risk. The clinician should assess the patient's preoperative risk factors and the risks associated with the planned surgery. It is often helpful to give an estimate of the percentage risk of cardiac complications (see above, by risk class) so that the surgeon can make the most educated decision regarding whether or not to proceed with surgery. The decision to undergo further testing depends upon the interaction of the patient's risk factors, surgery-specific risk and functional capacity. If a major risk predictor is present, nonemergency surgery should be delayed for medical management, risk factor modification and possible coronary angiography. For patients at intermediate clinical risk, both the exercise tolerance and the extent of the surgery are taken into account with regard to the need for further testing**.**

Patient-Related Predictors for Risk of Perioperative Cardiac Complications

Major clinical predictors (markers of unstable coronary artery disease)
Myocardial infarction <6 weeks
Unstable or severe angina (class III-IV)
Decompensated congestive heart failure
Significant arrhythmias (e.g., causing hemodynamic instability)
Severe valvular disease (e.g., aortic or mitral stenosis with valve area <1.0 cm^2)
CABG or PTCA <6 weeks
Intermediate clinical predictors (markers of stable coronary artery disease)
Previous myocardial infarction >6 weeks and <3 months (>3 months if complicated) based on the history or the presence of pathologic Q waves
Mild angina (class I-II)
Silent ischemia (Holter monitoring)
Compensated congestive heart failure, ejection fraction <0.35
Post CABG or PTCA >6 weeks and <3 months, or >6 yr, or with anti-anginal therapy
Diabetes mellitus
Renal insufficiency
Minor clinical predictors (increased probability of coronary artery disease)
Familial history of coronary artery disease
Age >70 yr
ECG abnormalities (arrhythmia, LVH, left bundle branch block)
Low functional capacity
History of stroke
Uncontrolled systemic hypertension
Hypercholesterolemia
Smoking
Post infarction (>3 months), asymptomatic without treatment
Post CABG or PTCA >3 months and <6 yr, and no symptoms of angina nor anti-anginal therapy
CABG= coronary artery bypass grafting, PTCA= percutaneous transluminal coronary angioplasty, LVH=left ventricular hypertrophy

Assessment Summary

I . History

- *Respiratory system : smoking,URTI,suppurative lung diseas..etc*
- *CVS: HF,IHD,Dyshythermia,HT…ETC*
- *CNS : neurological & psychological diseases*
- *Abdominal : GIT & Renal*
- *Medical disease: anaemia DM ,endocrine diseases.*
- *Drug allergies*
- *Previous operations & its complications.*

II. O/E

- *General examinatione.g vital sigs*
- *Systemic evaluations*

III. *Lab. Investigations e.g.* CBP, ESR, RFT, FBS, GUE, Electrolytes (Na, K^+, $Ca^{+"}$)

Suggestive Reading

Norman S William, Roman O Connell, Andrew W McCaskie. Bailey & Love short practice of surgery, 27th edition. Taylor and Francis, 2018

General Anesthesia

General anaesthesia (anesthesia) is a medically induced coma with loss of protective reflexes, resulting from the administration of one or more general anaesthetic agents.

Stages of anaesthesia

Guedel's classification, introduced by Arthur Ernest Guedel in 1937 describes four stages of anaesthesia.

- Stage 1 : Also known as induction, is the period between the administration of induction agents and loss of consciousness. During this stage, the patient progresses from analgesia without amnesia to analgesia with amnesia. Patients can carry on a conversation at this time.
- Stage 2 : Also known as the excitement stage, is the period following loss of consciousness and marked by excited and delirious activity. During this stage, the patient's respiration and heart rate may become irregular. In addition, there may be uncontrolled movements, vomiting, suspension of breathing, and pupillary dilation. Because the combination of spastic movements, vomiting, and irregular respiration may compromise the patient'sairway, rapidly acting drugs are used to minimize time in this stage and reach Stage 3 as fast as possible
- Stage 3 : Also known as surgical anaesthesia, the skeletal muscles relax, vomiting stops, respiratory depression occurs, and eye movements slow and then stop. The patient is unconscious and ready for surgery.

  This stage is divided into four planes:
  - The eyes roll, then become fixed;
  - Corneal and laryngeal reflexes are lost;
  - The pupils dilate and light reflex is lost;
  - Intercostal paralysis and shallow abdominal respiration occur.
- Stage 4 :Also known as overdose, occurs when too much anaesthetic medication is given relative to the amount of surgical stimulation and the patient has severe brainstem or medullary depression, resulting in a cessation of respiration and potential cardiovascular collapse. This stage is lethal without cardiovascular and respiratory support.

The process of anesthesia

A. Premedication

Prior to administration of a general anaesthetic, the anaesthetist may administer one or more drugs that complement or improve the quality or safety of the anaesthetic. The aim of remedications which is administrated 1-2 hr prior operation are:

- *Analgesia*
- *Anxiolysis*
- *Increase in gastric PH and decrease of gastric secretion*
- *Decrease saliva secretion*

The commonly used premedication :

- Midazolam, a benzodiazepine characterized by a rapid onset and short duration, is effective in reducing preoperative anxiety
- Beta adrenergic antagonists to reduce the incidence of postoperative hypertension, cardiac dysrhythmia, or myocardial infarction
- Opiods e,g. fentanyl, pethidine as analgesics
- Antiemetics e,g. metoclopramide, Dexamethasone
- Medications which reduce gastric volume, increase gastric PH and decrease gastric motility (to reduce risk of aspiration):
- Antacids , i.e. H2 receptor antagonists e.g. Tagamet (cimetidine) and Zantac (ranitidine hydrochloride) and Proton pump inhibitors e.g. Omeprazole

B. Induction

General anaesthesia is usually induced in a medical facility, most commonly in an operating theatre. Anaesthetic agents may be administered by various routes,including inhalation, injection (intravenous, intramuscular, subcutaneous), oral, and rectal. Most general anaesthetics are induced either intravenously or by inhalation. Intravenous injection works faster than inhalation, taking about 10–20 seconds to induce total unconsciousness. This minimizes the excitatory phase (Stage 2) and thus reduces complications related to the induction of anaesthesia.

- Commonly used intravenous induction agents include propofol, sodium thiopental, etomidate, methohexital, and ketamine.
- Inhalational anaesthesia (including Sevoflurane, Desflurane, Isoflurane, Enflurane, Halothane) may be chosen when intravenous access is difficult to obtain (e.g., children), when difficulty maintaining the airway

is anticipated, or when the patient prefers it. Sevoflurane is the most commonly used agent for inhalational induction, because it is less irritating to the tracheobronchial tree than other agents.

Anaesthetized patients lose protective airway reflexes (such as coughing), airway patency, and sometimes a regular breathing pattern due to the effects of anaesthetics, opioids, or muscle relaxants. To maintain an open airway and regulate breathing, some form of breathing tube is inserted after the patient is unconscious. To enable mechanical ventilation, an endotracheal tube is often used.

Other drugs used in general anesthesia

- Muscle relaxants: drug that affects skeletal muscle function and decreases the muscle tone. Useful for aiding insertion of Endotracheal tube and ventilation. There are two types of Neuromuscular blocking agents:
  i. Depolarising muscle relaxants Suxamethonium
  ii. Non-depolarising muscle relaxants ex, Atracurium
- Benzodiazepines: enhance the effect of the neurotransmitter gamma-aminobutyric acid (GABA) at the GABAA receptor, resulting in sedative, hypnotic (sleep-inducing), anxiolytic (anti-anxiety), anticonvulsant, and muscle relaxant properties. Ex: diazepam, midazolam, Lorazepam.
- Opioids: While opioids can produce unconsciousness, they do so unreliably and with significant side effects. So, while they are rarely used to induce anesthesia, they are frequently used along with other agents such as intravenous non-opioid anesthetics or inhalational anesthetics.Furthermore, they are used to relieve pain of patients before, during, or after surgery.Ex: Alfentanil, fentanyl

C. Maintenance

The duration of action of intravenous induction agents is generally 5 to 10 minutes, after which spontaneous recovery of consciousness will occur. In order to prolong unconsciousness for the required duration (usually the duration of surgery), anaesthesia must be maintained. This is achieved by allowing the patient to breathe a carefully controlled mixture of oxygen, sometimes nitrous oxide, and a volatile anaesthetic agent, or by administering medication (usually propofol) through an intravenous catheter.

D. Emergence

Emergence is the return to baseline physiologic function of all organ systems after the cessation of general anaesthetics. This stage may be

accompanied by temporary neurologic phenomena, such as agitated emergence (acute mental confusion), aphasia (impaired production of speech), or focal impairment in sensory or motor function.Shivering is also fairly common and can be clinically significant because it causes an increase in oxygen consumption.

Reversal of anesthetics by

- ✓ Switch off Inhalational /Intravenous Anaesthetic agents
- ✓ Using Intravenous reversal agents

i. Flumazenil, reverses the effects of benzodiazepines

ii. Naloxone, reverses the effects of opioids

iii. Neostigmine, helps reverse the effects of non-depolarizing muscle relaxants

Causes of postponing surgery

1. Not well controlled medical dis. E.g. Htn, DM, anemia.
2. Not well resuscitated pt. (traumatic pt. with uncontrolled acute bl. loss).
3. Acute URTI.
4. Recent ingestion of food. (<5 hrs).
5. Failure to get concept or guideline from the pt.

Postoperative complications

- Pulmonary complications (inadequate respirations, Atelectasis, Pneumonia, Pneumothorax)
- CVS complications (Hypotension, Dysrrhythmias , MI, DVT)
- CNS complications (central and peripheral problems)
- Abdominal complications (hepatic, renal)
- Local (haematoma and thrombophilibitis)
- Nausea & vomiting,
- Sore throat.
- Hoarseness.
- Laryngeal granuloma.
- Headache.

- Trauma to the teeth.
- Ocular Complications.
- Post-op. Parotitis.
- Wound complications (Wound dehiscence, Wound Hge, hematoma & seroma, Wound infection, Hypertrophic or keloid scar.),

Suggestive Reading

Norman S William, Roman O Connell, Andrew W McCaskie. Bailey & Love short practice of surgery, 27th edition. Taylor and Francis, 2018

Metabolic Response to Trauma

Response to trauma includes various endocrine, metabolic and immunological changes. The severity of these changes is related to the amount of exposed stress. In the activation of central nervous system and hormonal responses against injury, the direct effect of mediators like TNF-α and IL 1, which are released from traumatic tissue, on the hypothalamus has been well-known. However, many new studies refer to nuclear factor kappa B (NF-kB) in this regard. In a burn-rat model study, it was stated that melatonin, which is protective against liver damage, played a role in the suppression of NF-kB that is accepted as a mediator of inflammatory response, and melatonin treatment reduced the significantly increased hepatic NF-kB and TNF-α activity .

Stress response caused by events such as surgical trauma includes endocrine, metabolic and immunological changes. Stress hormones and cytokines play a role in these reactions. More reactions are induced by greater stress, ultimately leading to greater catabolic effects. Authors reported the characteristic response that occurs in trauma patients: protein and fat consumption and protection of body fluids and electrolytes because of hypermetabolism in the early period. The oxygen and energy requirement increases in proportion to the severity of trauma. The awareness of alterations in amino acid, lipid, and carbohydrate metabolism changes in surgical patients is important in determining metabolic and nutritional support. The main metabolic change in response to injury that leads to a series of reactions is the reduction of the normal anabolic effect of insulin, i.e. the development of insulin resistance. Free fatty acids are primary sources of energy after trauma. Triglycerides meet 50 to 80 % of the consumed energy after trauma and in critical illness. Surgical stress and trauma result in a reduction in protein synthesis and moderate protein degradation. Severe trauma, burns and sepsis result in increased protein degradation. The aim of glucose administration to surgical patients during

fasting is to reduce proteolysis and to prevent loss of muscle mass. In major stress such as sepsis and trauma, it is important both to reduce the catabolic response that is the key to faster healing after surgery and to obtain a balanced metabolism in the shortest possible time with minimum loss. For these reasons, the details of metabolic response to trauma should be known in managing these situations and patients should be treated accordingly.

It is a natural physiological response to any kind of injury, mediated by various neural & hormonal reflexes (centers within hypothalamus & is effective via sympathetic neuron system).It is divided into:

1- Early phase. **2**- Catabolic phase. **3**- Turning phase. **4**- Anabolic phase.

Early phase : It is temporary phase ,decrease metabolic rate ,decrease body temperature., increase blood pressure, increase heart rate , Increase output of catecholamines from adrenal medulla ,Increase glycogenolysis in the liver, Increase lactic acid released from muscles ,increase plasma level of fatty acids. Decrease insulin secretion which promotes gluconeogensis in the liver and hyperglycemia.

Catabolic phase: excessive secretion of Corticosteroids like cortisol & aldosteron from adrenal cortex & ADH from post, pituitary, characterized by metabolic, fluid & electrolyte changes

Turning phase: i.e. CS withdrawal (K^+ balance return to normal , K^+ return inside the cells and Na^+ excreted with H_2O. , Diuresis with increase U.O. and +ve N_2 balance ,N_2 loss stops & proteins synthesis starts .

Anabolic phase: No more cortisol secretion, Growth Hormone & insulin secretion , and androgen &estrogen secretion ,+ve N2 balance, Re-synthesis of muscle proteins and in turn increase Body weight .

Factors Affecting Surgical Response

- Age: Surgically induced metabolic and endocrine responses are usually different in children than in adults . Differences even between term and preterm neonates have been identified. As age increases, the hormonal response in the postoperative period lasts longer.
- Nutrition and diet: Perioperative nutritional status and especially the degree of diet affect the metabolic response to surgery. Postoperative metabolic response is increased by preoperative nutritional support. Patients who received nutritional support for prolonged periods have more postoperative insulin resistance. The type of fluid given intraoperatively also affects the metabolic response directly or indirectly.
- Anesthesia: The type of anesthesia also affects surgical stress response. Both general and local/regional anesthesia has been used to reduce the inflammatory response to surgery. Some authors suggested that mortality was reduced in newborn infants with major cardiac surgery by reduction of metabolic response with administration of deep anesthesia and postoperative analgesia. It was reported in a randomized controlled trial that endocrine response to surgery and postoperative complications were reduced in preterm with the addition of fentanyl to general anesthesia. Epidural block with local anesthetic agents particularly alters the metabolic response to surgical stress. Epidural block significantly decreases protein degradation without affecting whole body protein synthesis in adults, within the first 24 hours after surgery. Epidural block with bupivacain has no effect on protein, carbohydrate, or lipid metabolism when a surgical procedure is not performed. Epidural block alters postoperative response rather than directly affecting the metabolism.
- Surgical method: Insufflation of the abdominal cavity with CO2 or other gases affect response to metabolism. It is important to define metabolic changes associated with CO2 pneumoperitoneum. CO2 insufflation can cause both local and systemic responses affecting metabolic response to surgery. As shown in the

example of cholecystectomy, metabolic response is less common in minimally invasive surgery. Therefore, it caused less tissue trauma and less inflammatory response. Cytokine synthesis from mesothelial cells were less after laparoscopy as compared to open surgery.

- Operative stress: Surgical trauma/stress level is one of the factors that affect the magnitude of inflammatory and metabolic response to surgery. The metabolic response to trauma was also confirmed in infants and children with new findings.
- Intraoperative and postoperative thermoregulation in response to surgery: Intraoperative thermoregulation is one of the main determinants of metabolic response. Changes in thermoregulation also play an important role in determining the postoperative metabolic response. Thermoregulation varies intraoperatively depending on effects of anesthetic drugs, opened body cavities and loss of most of the normal regulatory control mechanisms. Anatomical and physiological differences in thermoregulation of the newborn, child and adult are partly responsible for the different patterns of postoperative metabolic response.

Suggestive Reading

Norman S William, Roman O Connell, Andrew W McCaskie. Bailey & Love short practice of surgery, 27th edition. Taylor and Francis, 2018

Hemorrhage

Classification & Types

- **Arterial Hemorrhage**: it is recognized as bright red blood, spurting as a jet which rises and falls in time with pulse.
- **Venous Hemorrhage**: it darker red steady and copious flow, the color dark from excessive oxygen desaturation, later it becomes more darker because of further bleeding, except venous bleeding from pulmonary veins which is bright red due to oxygenation.
- **Capillary Hemorrhage**: it is bright red, often rapid ooze.
- **Primary Hemorrhage**: occur at the time of injury or RTA immediately or seen in surgical incisions at operation.
- **Reactionary Hemorrhage**: it is occur usually within 4-6 hrs after injury or operation, but in general it is with first 24 hrs, mainly to slipped ligature, or knot are not strong enough o dislodgement of blood clot, or post operative increase in blood pressure, or even post operative increase in venous blood pressure.
- **Secondary Hemorrhage**: it occur mainly 1-7 or even to 14 days post operatively, due to infection, erosion of blood vessel, CA affect blood vessel wall, bone fragment or even metal fragment that cause pressure and irritation to blood vessel,< mainly appear and start as little bleeding few days after operation on dry wound dressing.
- **Internal Hemorrhage**: that not seen by eye e.g. ruptures spleen or liver with bleeding in the peritoneal cavity or cerebral hemorrhage.
- **External Hemorrhage**: can be seen by eye e.g. penetration of skin by knife or piece of glass or bullet, or haematomesis, haematuria, vaginal bleeding…etc.

Clinical Presentation of Patient with Hemorrhage

1. Pale, cold wet skin.
2. Restlessness, anxiety mainly due to effects of hypoxia on midbrain.
3. Rapid pulse with normal or increase BP which is maintained first by compensatory mechanisms when the bleeding continues or becomes severe leading to hypotension.
4. Deep sighing breathing (air hunger).
5. Thirst, occur in late stages.
6. Empty veins & difficult IV cannulation

Clinical & Lab. Measures

1. Charting of the patient vital signs (PR,RR,BP, TEMP.) should be done
2. monitoring of patient clinical signs.(polar, sweating cyanosis, cold nose…etc)
3. Blood loss assessments (clinical judgments depend on clinical observation and experience e.g. weighing of dressing or packs, blood clot size, sucker volume…etc)
4. PCV & Hb testing (those should be repeated every 8 hrs for accuracy especially in case of blood loss and after blood transfusions.
5. Central venous pressure measurement, this can be done by introducing a catheter through the arm veins till it reach superior vena cava (SVC) and this will give the readings on a manometer.

Treatment of Bleeding

- **Stop of bleeding**

1. Applying of pressure or compression by bandage or sterile gauze on stie of bleeding.
2. Digital pressure directly on bleeding area or site.
3. Packing by means or rolls of gauze or cotton.
4. Use of tourniquet (less than 1 hrs)
5. Position and rest (e.g., limb elevation …etc)

6. Vascular control (proximal & distal to the operation field and site.
7. Spry coagulation using Argon beam system for surface oozing over a wide area.
8. Sedation e.g. diazepam 10mg……morphine 15-20mg….pethidine 100mg.
9. Cautery and diathermy (thermal application)
10. Cryotherpy (thermal application)
11. Ligatures.
12. Surgical clips.
13. Surgical operation e.g. spleenectomy
14. drugs administration e.g Adrenaline
15. bone wax
16. synthetic substances e.g. gelfoamor oxycell
17. Recently Laser applications

- **Restore of blood volume**: mainly by blood transfusion, IV fluids(dextran, Macrodex and normal saline) ,platelets or FFP.

Suggestive Reading

Norman S William, Roman O Connell, Andrew W McCaskie. Bailey & Love short practice of surgery, 27th edition. Taylor and Francis, 2018

Case history and clinical examination

Dr. Sabah Alheeti

The art of taking accurate case history is probably the most important single step in the diagnosis of a medical or surgical condition.
A case history may be divided into:

I. The patient name, age, occupation and address
II. Chief complaint
III. History of present illness
IV. Medical, social, dental and family history.

- Cc: The nature and duration of the presenting symptoms should be considered briefly in one- or two-word summary. abdominal pain, nausea and vomiting.

- HPI: what was the first thing that he noticed wrong?
what other symptoms have occurred?
what make it better or worse?
what seems to be the main trouble now?
what treatment has he had and does it help?
what does the patient think he is suffering from?
ask about: (appetite, weight, bowl habits, sleep, dypnea, chest pain or swollen ankles).

-Medical Hx: inquire of the patient what diseases, operations or accidents he has sustained and list them in chronological order .Always give the dates and do not write 'three years ago' ….ect.
-Family Hx: Health and medical status of patient relatives should be asked.
-Social history: this part of history enables the physician to build up a picture of patient background like smoking, drinking alcohol and occupation.

Clinical examination:

After accurate case history has been taken, the clinical examination is carried out. This consists of:-
1- General physical examination of the patient using the principles of examination (inspection, palpation, percussion, auscultation).
2- Local examination of the lesion which carefully elicits all its clinical characteristics.

Clinical examination of patient with a pain

Pain anywhere should have the same features elicited. These can be summarized by the acronym SOCRATES.

- ❑ Site :where is the pain, is it localized or generalized?
- ❑ Onset : Gradual or sudden? Intermittent or generalized?
- ❑ Character: Sharp, stabbing, dull, aching, sore?
- ❑ Radiation : Does it spread to other areas?(from loin to groin in ureteric pain, to jaw and neck in myocardial pain)
- ❑ Associated symptoms: Nausea, vomiting, dysuria, jaundice?
- ❑ Timing: Does it occur at any particular time?
- ❑ Exacerbating or relieving factors: relief with hot water bottles suggest deep inflammatory or infiltrative pain.
- ❑ Surgical history: Does the pain relate to surgical intervention.

- **Other common surgical symptoms:** dyspepsia, dyspnoea, dysphagia, haematemesis, haemoptysis, abdominal distension, jaundice, change in bowel habit

The examination of lump

Before carrying out a local physical examination of any lump or mass, it is essential to ascertain:-

- How long the swelling has been present?
- Whether it is getting larger?
- Whether there is any possible cause for swelling., trauma.

The features of lump that should be considered can be remembered by acronym (4 students and 3 teachers around the campfire):-

- Site: the lump may arise from skin, s.c. , muscle, tendon, BV., nerves or organ. The lump must be described with reference to the body surface landmarks,e.g. angle of mandible.
- Size
- Shape
- Surface: of the mass may be smooth, lobulated or irregular.
- Tenderness: on gentle palpation is valuable physical sign. Inflammatory lumps are tender while neoplasms are painless unless secondarily infected.
- Temperature: the site of acute inflammation is usually warmer than the adjoining areas.
- Transillumination: Whether a torch behind lump will allow light to shine through the lump. The only readily transilluminable swelling of the head and neck is the cystic hygroma .
- Consistency and color: the consistency of lump is defined surgically as Soft as in lipoma , Firm as in fibroma , Cartilage Hard as in pleomorphic adenoma, Bony hard as in osteoma, Rock hard as in malignant lymph nodes, Rubbery hard as in Hodgkin lymphoma.

The color of lump may be helpful diagnostic sign like reddening may suggest inflammatory etiology.

- Appearance of patient: massive swellings associated with cachexia of the patient are usually indicative of malignant neoplasms.
- Mobility: Move lump in two directions, right-angled to each other. Then repeat exam when muscle contracted:

a) Bone: immobile.
b) Muscle: contraction reduces lump mobility.
c) Subcutaneous: skin can move over lump.
d) Skin: moves with skin

- Pulsation: Assess with 2 fingers on mass, there are three types of pulsation which may occur in lumps:

a) The mass may be pulsatile: aneurysm
b) Transmitted pulsation occur when the mass rests on a large artery,e.g. palatal adenoma of the palate which transmit pulsation of greater palatine artery.
c) A mass lying deep in the tissue may displace artery so that it lies superficially upon the mass.

- Fluctuation: indicate presence of fluid within the lump. It is elicited by placing the tips of two fingers on the lump. When pressure is applied to the mass with one finger, transmitted upward impulse is felt with the other finger-tip.
- Reducibility: Reducible mass reappears only on cough,e.g. hernia.
- Regional lymph nodes
- Edge: of the lump may be clearly defined or diffuse, fading into the surrounding tissues as in inflammatory lumps.

Examination of ulcer

Is a discontinuity or break in a skin or mucous membrane.

Classification:

1. Venous
2. Arterial
3. Diabetic
4. Neuropathic
5. Traumatic
6. Malignant
7. Infective

local examination of an ulcer:

A) *Inspection*: we should note

1) Size & Shape (ulcer may be round, oval, crescentic, irregular in shape)

2) Number

3) Location: many ulcers occur in characteristic sites.

- Varicose ulcer → medial aspect of lower third of the leg
- Rodent ulcer → nose
- Squamous cell carcinoma ulcer ⟶ tongue
- Tuberculous ulcer → neck
- Syphilis ⟶ junction of hard and soft palate
- Trophic ulcer → weight-bearing area (e.g. heel of the feet)
- Bedsore ulcer→ sacrum

4) Floor: is the exposed surface of the ulcer. We should note:-

- ❖ The granulation tissue (this may be red, pale), amount of sloughing (necrotic tissue not yet separated from living tissue), membrane..ect.
- ❖ Discharge: which may be
- ❑ Serous (plasma that's thin, clear and watery),
- ❑ Serosanginous (This leakage is thin and watery, and it's pink in color (it can also be a darker red),
- ❑ Sanginous (fresh blood)
- ❑ Purulent. (gray, green or yellow, and purulent drainage is most commonly thick in consistency)

5) Margin & Edge: ***Margin*** *is the border or transitional zone of skin around an ulcer*. There are three types:

-Healing margin [white (outer) – blue (central) – red (Inner)]

-Inflamed margin (red, irregular margin with inflamed surrounding skin)

-Fibrosed margin (thickened white)

Edge *is the mode of union between the floor and the margin of* ulcer. There are five types: (see figure below)

o Sloping edge → healing ulcer

o Punched edge → trophic ulcer, syphilis

o Undermined edge → tuberculous ulcer

o Everted edge→ malignant ulcer

o Raised (rolled) edge → basal cell carcinoma (rodent ulcer).

6) Surrounding skin: if ulcer is spreading and infected the surrounding skin is shiny, red, edematous due to cellulitis.

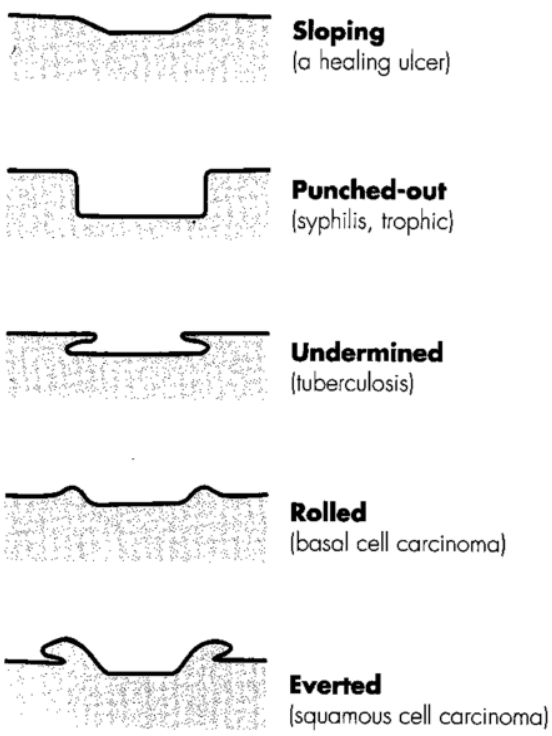


FIG 1.15 The varieties of ulcer edge.

B)Paplation

1) Surrounding skin for temperature and tenderness.
2) Ulcer: edge, base
3) Test the fixity of the ulcer to the structures in its base.

Base (tissue on which the ulcer rests): note
• Consistency (soft, indurated or fixed)
• Underlying structures (muscle, fascia or bone).

C) Focal examination

1. Regional Lymph node,e.g.
• Hard, discrete, non-tender → malignant ulcer
• Soft, tender → infective
• Non-tender, matted → tuberculous ulcer
2. State of arteries, venous circulation, nerves
3. Movement of neighboring joints

D) Systemic examination

• Cardiovascular: for CHF which delays ulcer healing
• Respiratory: for TB .

Fluid and electrolyte management

Dr. Sabah Alheeti

Learning objective

- To understand the distribution and composition of body fluids, and how these may change following surgery
- To understand types of intravenous fluid therapy and common electrolyte disorders

The management of a patient' s fluid status is vital to a successful outcome in surgery. This requires preoperative assessment, with resuscitation if required, and postoperative replacement of normal and abnormal losses until the patient can resume a normal diet.

Body fluid compartments (Figure 1)

In the 'average' person, water contributes 60% to the total body weight: 42 L for a 70 kg man. 40% of the body weight is intracellular fluid, while the remaining 20% is extracellular. This extracellular fluid can be subdivided into intravascular (5%) and extravascular, or interstitial (15%). Fluid may cross from compartment to compartment by osmosis, which depends on a solute gradient, and filtration, which is the result of a hydrostatic pressure gradient.

The electrolyte composition of each compartment differs. Intracellular fluid has a low sodium and a high potassium concentration. In contrast, extracellular fluid (intravascular and interstitial) has a high sodium and low potassium concentration. Only 2% of the total body potassium is in the extracellular fluid. There is also a difference in protein concentration within the extracellular compartment, with the interstitial fluid having a very low concentration compared with the high protein concentration of the intravascular compartment.

Knowledge of fluid compartments and their composition becomes very important when considering fluid replacement. In order to fill the intravascular compartment rapidly, a plasma substitute or blood is the fluid of choice. Such fluids, with high colloid osmotic potential, remain within the intravascular space, in contrast to a saline solution, which rapidly distributes over the entire extravascular compartment, which is four times as large as the intravascular compartment. Thus, of the original 1 L of saline, only 250 mL would remain in the intravascular compartment.

Osmolality refers to the number of osmoles of solute particles per kilogram of water. The asymmetric accumulation of effective osmoles in either extracellular fluid (e.g., Na+, glucose, mannitol, and glycine) or intracellular fluid (e.g., K+, amino acids, and organic acids) causes transcompartmental movement of water. Because the cell membrane is freely permeable to water, the osmolalities of the extracellular and intracellular compartments are equal. The effective osmolality of a solution is equivalent to its tonicity.

Plasma osmolality= 2[Na+] + [Glucose]/18 + [BUN]/2.8

- The normal range is 280-290 mOsm/L

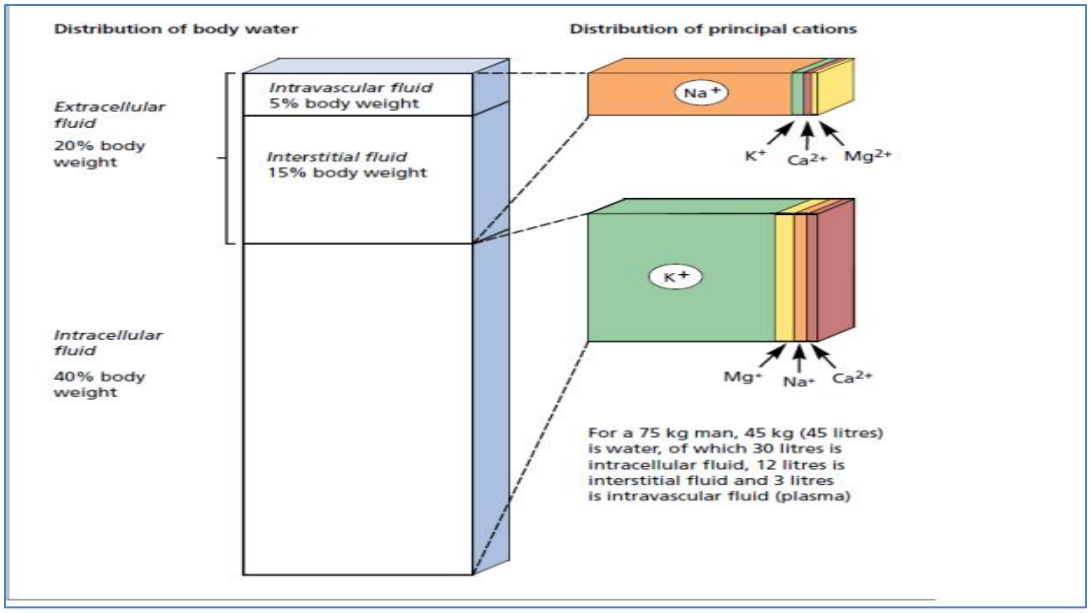


Figure 1: Distribution of fluid and electrolyte in body

Table 2.1 Normal daily fluid losses

Fluid loss	Volume (mL)	Na^+ (mmol)	K^+ (mmol)
Urine	2000	80–130	60
Faeces	300		
Insensible	400		
Total	2700		

Types of intravenous fluids

The fluids used in clinical practice are usefully classified into colloids and crystalloids.

1-Colloids: solutions that contain large molecules that can't pass through gap junctions of blood vessels. When infused, they remain in the intravascular compartment and expand it and they draw fluid from extravascular spaces via their higher oncotic pressure.

Indications:

- Rapid replacement of intravascular fluid (so called plasma expander)
- Correct albumin and protein level

Examples: albumin, plasma, blood and its products, dextran.

2- Crystalloids: solutions contain small molecules that flow easily across cell membranes, allowing for transfer from the bloodstream to the cells and tissues. This will increase fluid volume in both the interstitial and intravascular spaces.

It is divided according to osmolarity into:

- Isotonic : 0.9% NaCl, lactate ringer , 5% dextrose
- Hypotonic: 0.45%NaCl
- Hypertonic: D5 in 0.9% normal saline.

Intravenous infusion	Na^+ (mmol/L)	Cl^- (mmol/L)	K^+ (mmol/L)	HCO_3^- (mmol/L)	Ca^{2+} (mmol/L)
Normal saline (0.9% saline)	150	150	–	–	–
4% dextrose/ 0.18% saline	30	30	–	–	–
Hartmann's (compound sodium lactate)	131	111	5	29	2
Normal plasma values	134–144	95–105	3.4–5.0	22–30	2.2–2.6

INDICATIONS OF FLUID THERAPY

I. Fluid resuscitation

Patients who are in hypovolemic shock require rapid fluid infusions in the form of fluid challenges to maintain intravascular volume.
Rapid infusion of a 1000 mL bolus of normal (isotonic) saline (NS) or lactated Ringer's solution (RL) within 15 minutes.

II. Replacement of ongoing fluid loss

Fluids are also indicated in the post-resuscitation phase when the patient is no longer hypovolemic but still has ongoing abnormal fluid loss that cannot be compensated for by oral intake alone.
Some common conditions associated with an ongoing fluid loss are:

- Burns
- Polyuria (high output renal failure, diabetes insipidus)
- Surgical drainage
- Significant ongoing gastrointestinal loss (vomiting, diarrhea)

III. Maintenance fluid therapy

Maintenance fluids are indicated in patients who cannot or are not allowed to take fluids orally.
The most commonly used formula is

Holliday-segar method which is 4-2-1 or 100-50-20.

- 100 ml/kg/24-hours = 4 ml/kg/hr for the 1st 10 kg
- 50 ml/kg/24-hours = 2 ml/kg/hr for the 2nd 10 kg
- 20 ml/kg/24-hours = 1 ml/kg/hr for the remainder of weight

Example: 70-kg patient with severe mandibular fracture and can not take fluids orally. What is the amount of maintenance fluids which should be administrated to patient in 24 hrs?
100 x 10 kg= 1000 ml
50 x 10 kg= 500 ml
The remainder of 70kg is 50 kg, so 50kg x 20=1000 ml
The Total is (2500 ml) which is the amount of maintenance fluid.

Abnormalities of body water

1-Dehydration: is excessive loss of body water.

- Causes:
  I. Insufficient fluid intake
  II. Excess loss of fluid (surgical procedure, diseases)
  III. Haemorrhage
  IV. Diarrhea, bowel obstruction, vomiting
  V. Fistula
  VI. Diuresis by drugs
  VII. Insensible losses (pyrexia)
- Signs and symptoms:
  I. Dry mouth, thirst, oliguria
  II. Decreased skin turgor, pinched face, tachycardia, postural hypotension
- Management:
  I. Identify source of sodium loss if present because loss of water also lead to hyponatremia
  II. Mild- moderate dehydration: use oral rehydration solutions
  III. Severe dehydration: use i.v. isotonic solution such as N/S.

2-Fluid overload and oedema

Excess body water occurs in several diseases

- Causes:
  I. Excess intake of fluid: most common cause, ex. Excess i.v. fluid administration
  II. Decreased loss of water with salt retention which may occur in many diseases such as renal failure, liver failure, cardiac failure.
- Signs and symptoms:
  I. Oedema
  II. Weight gain
  III. Orthopnea
  IV. Increase blood pressure
  V. Distention of jugular vein
- Management
  I. Limit sodium intake
  II. Use of diuretics like frusemide

Head injuries

Dr.sabah alheeti

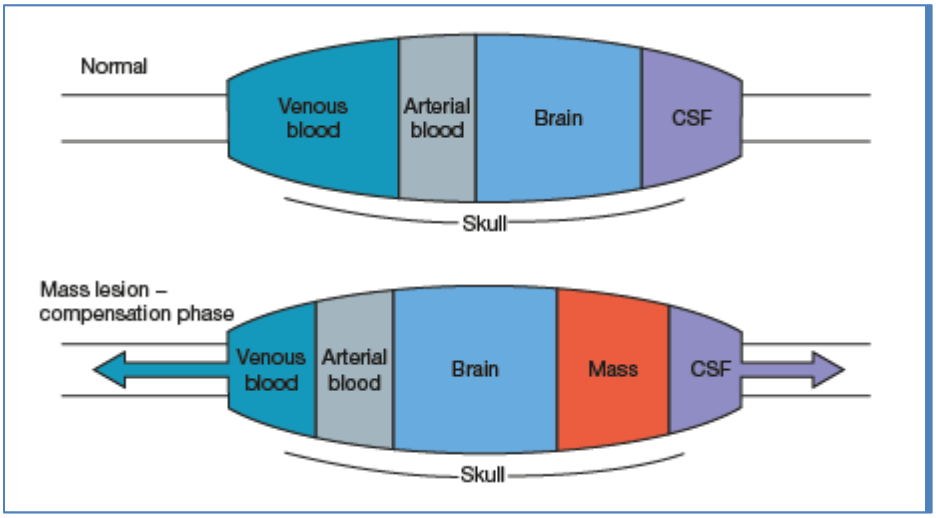
Head injury accounts for 3–4% of emergency department attendances. Head injury is Any injury that results in trauma to the scalp, skull or brain. Head injury and traumatic brain injury are often used interchangeably.

Intracranial pressure and cerebral blood flow

The brain depends on continuous perfusion for oxygen and glucose delivery, and hence survival. Normal cerebral blood flow (CBF) is about 55 mL per minute for every 100 grams of brain tissue. Ischaemia results when this rate drops below 20 mL per minute, and even lower levels will result in infarction unless promptly corrected. Flow depends on cerebral perfusion pressure (CPP), the difference between mean arterial pressure (MAP) and intracranial pressure (ICP).

CPP (75–105 mmHg) **= MAP** (90–110 mmHg) **– ICP** (5–15 mmHg)

Alexander Monro observed in 1783 that the cranium is a 'rigid box' containing a 'nearly incompressible brain'. Any expansion in the contents, especially haematoma and brain swelling, may be initially accommodated by exclusion of fluid components, venous blood and cerebrospinal fluid (CSF). Further expansion is associated with an exponential rise in ICP.
The Monro-Kellie doctrine or hypothesis states that the sum of volumes of brain, cerebrospinal fluid (CSF) and intracerebral blood is constant. An increase in one should cause a reciprocal decrease in either one or both of the remaining two to maintain equilibrium and sustain a normal ICP.



CLASSIFICATION OF HEAD INJURY

1. **According to GCS:**

Table 18-1 Glasgow Coma Scale

Action	*Score*
Eye opening	
Spontaneously	4
To speech	3
To pain	2
None	1
Motor response	
Obeys	6
Localizes pain	5
Withdraws from pain	4
Flexion to pain	3
Extension to pain	2
None	1
Verbal response	
Oriented	5
Confused	4
Inappropriate	3
Incomprehensible	2
None	1

Adapted from Teasdale G and Jennett B.[9]
Patient's score determines category of neurologic impairment: 15 = normal; 13 or 14 = mild injury; 9–12 = moderate injury; 3–8 = severe injury.

2. **According to mechanism of production, head injury can be classified as:**
   - Impact injuries: It results from an object striking the head or the head striking an object. It includes: Scalp injuries, Skull fracture, brain.
   - Acceleration and deceleration injuries: It results essentially as a result of differential movement between skull and cranial content. It includes: Diffuse axonal injury Subdural hematoma
   - Coup injury and Countre-coup injury:
     Coup injury: It occurs at the site of the impact to the head and is produced by compression of brain due to inward movement of the bone.
     Countre-coup: Injury occurs directly opposite to the point of impact and are most common in frontal and temporal lobe.

Consequences of head injury

1. **Injury to the scalp:** There can be scalp contusion, abrasion and/or lacerations

   - Profuse bleeding due to rich vascularity of scalp.
   - Pott's puffy tumor, first described by Sir Percivall Pott in 1760, is a rare clinical entity characterized by subperiosteal abscess associated with osteomyelitis of the frontal bone.

2. **Skull Fracture**: which may be

   - Simple linear fracture: It is the break in the bone that transverses the full thickness of the skull from the outer to inner table. They are usually no bone displacement.
   - Depressed Skull Fracture : are comminuted fractures in which broken bones displace inward. Compound depressed skull fractures occur when there is a laceration over the fracture, putting the internal cranial cavity in contact with the outside environment, increasing the risk of contamination and infection. In complex depressed fractures, the dura mater is torn. Depressed skull fractures may require surgery to lift the bones off the brain if they are pressing on it by making burr holes on the adjacent normal skull.
   - Basilar fractures: linear fractures that occur in the floor of the cranial vault (skull base), which require more force to cause than other areas of the neurocranium. Thus they are rare, occurring as the only fracture in only 4% of severe head injury patients.
     Basilar fractures have characteristic signs: **blood in the sinuses**; cerebrospinal fluid **rhinorrhea** (CSF leaking from the nose) or from the ears (**cerebrospinal fluid otorrhea**); periorbital ecchymosis often called **'raccoon eyes'** and retroauricular ecchymosis known as "Battle's sign" (bruising over the mastoid process)

3. Brain injury: which may be
   - Primary brain injury: Injury caused at the time of impact . it is Irreversible
   - Secondary brain injury: Subsequent or progressive brain damage occurs hours or days after primary brain injury.

Primary brain injury	*Secondary brain injury*
Concussion	Intracranial haematoma
Cortical laceration/contusion	Cerebral oedema
Diffuse axonal injury	Ischaemia
Bone fragmentation	Infection

Concussion

It is the condition of temporary dysfunction of brain without any structural damage following head injury.

It is manifested as: Transient loss of consciousness, Transient loss of memory, Autonomic dysfunction like bradycardia, hypotension and sweating.

Contusion

It is more severe degree of brain injury manifested by areas of hemorrhage in the brain parenchyma but without surface laceration. Neurological deficit persists more than 24 hour and associated with cerebral edema and defects in the blood brain barrier.

Diffuse axonal injury

DAI is a form of traumatic brain injury which results from mechanical shearing at grey-white interface due to severe acceleration and deceleration force. No obvious structural damage. Severity may range from mild damage with confusion to coma and even death.

Intracranial Hemorrhage

Intracranial hemorrhage encompasses four broad types of hemorrhage:

- epidural hemorrhage
- subdural hemorrhage
- subarachnoid hemorrhage
- intraparenchymal hemorrhage

Extradural haematoma

Collection of blood between the cranial bones and duramater
Can follow relatively minor trauma with brief loss of consciousness is typical.
Results from damage to middle meningeal artery
Followed by a lucid interval and then sudden deterioration
lens-shaped or biconvex lesion on computed tomography
Require immediate transfer to a neurosurgical unit for decision on evacuation.

Note: **lucid interval** is a temporary improvement in a patient's condition after a traumatic brain injury, after which the condition deteriorates

Subdural haematoma

Collection of blood between brain and dura mater
Acute: <3 days, Sub-acute: 4-21 days, Chronic: >21 days

- Results from torn bridging vein or injury to the cortical artery
- Haematoma extensive and diffuse

- No lucid interval
- Loss of consciousness occurs immediately after trauma and is progressive
- Features of raised ICP and focal neurological defecits
- CT Scan: Concavo-convex lesion
- T/t: surgical decompression by craniotomy + Antibiotics

Approach to Head Trauma

1.Detailed history should be sought in all cases of head trauma

2.Initial assessment of head injuries must follow advanced trauma and life support. (ALTS).

3. Neurological assessment: BY GCS, pupil size and reaction, checking neurological deficits such as paralysis, loss of sensation.

Treatment of raised ICP

- IV Mannitol
- IV furosemide
- Reverse Trendelenburg if no counter indications like hypovolaemia, spine injury
- If significant agitation and if hypoxia, hypovolaemia or pain is excluded as the cause of agitation: give IV Midazolam
- Analgesics for the pain management
- Phenytoin or phenobarbitone for post traumatic seizure

Nutritional Support

Dr. sabah Alheeti

Nutrition plays a vital role in the recovery of patients from surgery. It is estimated that between 30% and 50% of hospitalized patients are malnourished. Poor nutrition has deleterious effects on wound healing and immune function, which increases postoperative morbidity and mortality.

Nutritional requirements: The Calories are provided mainly by carbohydrate and fat

- Fat = 9 kcal/g
- Carbohydrate = 4 kcal/g
- Protein = 4 kcal/ g
- Daily caloric requirements: 30-35kcal/kg which is increased in sepsis, trauma, surgery or ventilation (35-40kcal/kg/day).
- Daily protein requirements in the average healthy adult without excessive losses are approximately 0.8 g/kg body weight

Malnutrition

According to the WHO, malnutrition is "the cellular imbalance between supply of nutrients and energy and the body's demand for them to ensure growth, maintenance, and specific functions.
What are the causes of malnutrition?

• Neglect (e.g., severe alcoholics, extreme of ages)
• Digestive problems
• Inadequate food intake
• Chronic illness
• Dysphagia
• Stress and trauma
• Vomiting

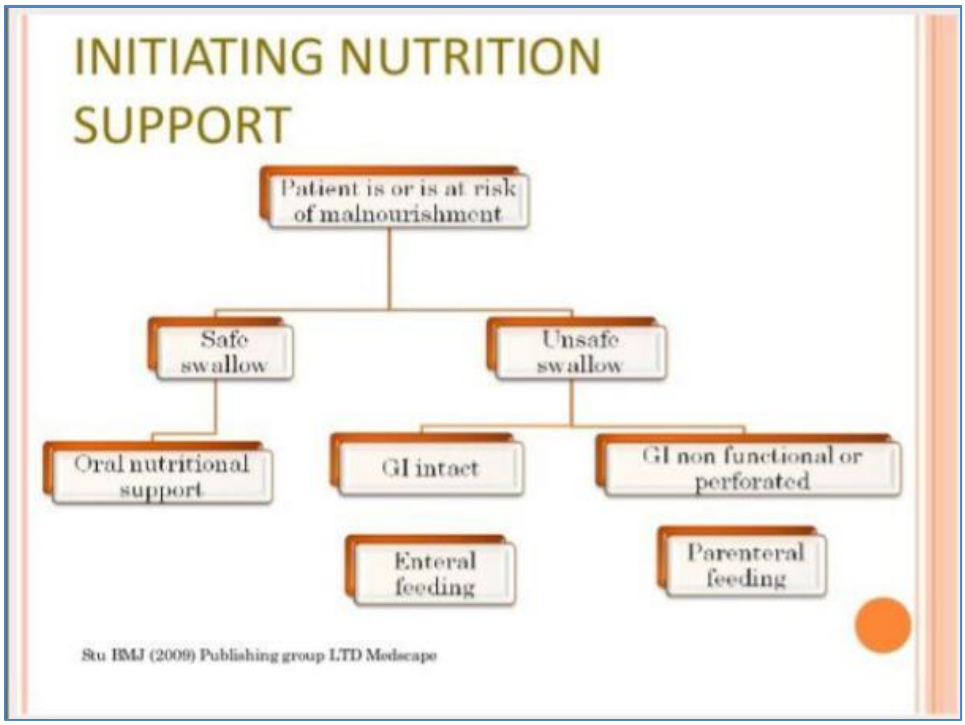
Nutritional assessment

1- Anthropometric measurements such as triceps skinfold thickness and midarm muscle circumference reflect body-fat stores and skeletal muscle mass, respectively.
2- body mass index (weight kg/height m2) < 18.5 indicates nutritional impairment.
3- Laboratory tests: Serum albumin of less than 3.5 g/dL, Serum prealbumin less than 5 mg/dl, Serum transferrin of less than 200 mg/dL.

Nutritional support is the provision of nutrients to patients who cannot meet their nutritional requirements by eating standard diets.

What are the indications for nutritional support?

- Inadequate intake for more than 5 days
- Malnourished patients undergoing surgery
- Major trauma (burn victims, blunt or penetrating injury, etc.)



Routes of nutritional support

1- Enteral nutrition
2- Parenteral nutrition

Enteral nutrition

- Delivery of nutrient into healthy and functioning gastrointestinal tract.
- Most preferred and more physiological
- Advantages:

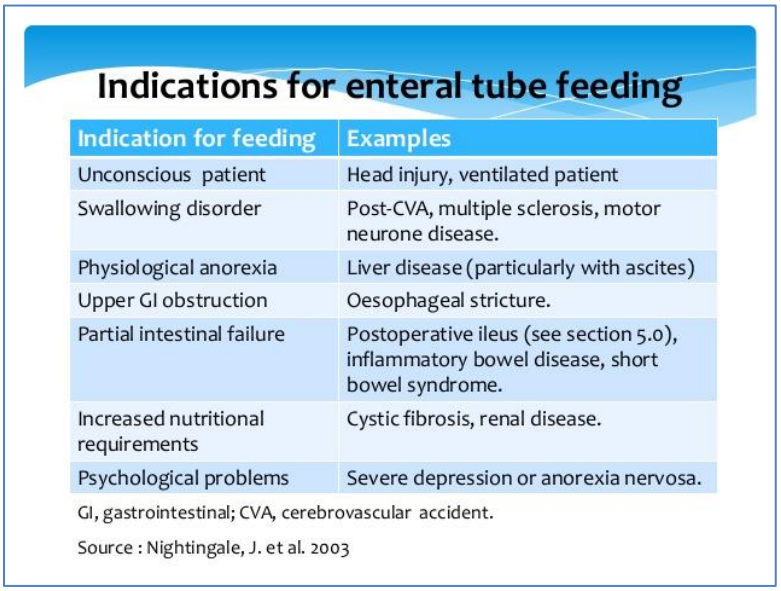
- Maintain gut mucosal integrity
- Maintain normal gut flora & pH
- Cheap & easily available
- Less complications

. Types of enteral feeding:

1) Nasogastric tube (or nasoduodenal or nasojejunal)
2) Gastrostomy : placing the feeding tube through abdominal wall
   Into stomach (surgically or endoscopic)
3) Jejunostomy : placing the feeding tube through abdominal wall
   Into jejunum (surgically or endoscopic)

- Note: For long-term enteral feeding, enterostomies are the preferred access route than nasogastric tube.
- Nasoduodenal tube feedings are indicated in:
  • Patients at risk for aspiration.
  • Patients who are debilitated, demented, stuporous, or unconscious.
  • Patients with gastroparesis or delayed gastric emptying.

- The available dietary formulations for enteral feedings can be divided into polymeric (blenderized and nutritionally complete commercial formulas), chemically defined formulas (elemental diets)
- Indication: Enteral feedings are indicated for patients who have a functional GI tract but are unable to sustain an adequate oral diet



Indications for enteral tube feeding

Indication for feeding	Examples
Unconscious patient	Head injury, ventilated patient
Swallowing disorder	Post-CVA, multiple sclerosis, motor neurone disease.
Physiological anorexia	Liver disease (particularly with ascites)
Upper GI obstruction	Oesophageal stricture.
Partial intestinal failure	Postoperative ileus (see section 5.0), inflammatory bowel disease, short bowel syndrome.
Increased nutritional requirements	Cystic fibrosis, renal disease.
Psychological problems	Severe depression or anorexia nervosa.

GI, gastrointestinal; CVA, cerebrovascular accident.

Source : Nightingale, J. et al. 2003

- Contraindication: intestinal obstruction, ileus, GI bleeding, severe diarrhea, vomiting, enterocolitis, or a high-output enterocutaneous fistula. It is also contraindicated in patients in shock.
- Complications:
  I. Tube-related: malposition, break, block and leakage of feeding tube.
  II. Gastrointestinal: nausea, vomiting, diarrhea and pulmonary aspiration
  III. Metabolic: constipation, electrolyte imbalance
  IV. Infections

Parenteral nutrition

Parenteral nutrition is a way to feed the patient intravenously. It can be delivered centrally through a central venous catheter, most commonly in the superior vena cava or peripherally (PPN) via a peripheral vein.
While the total Parenteral nutrition (TPN) is the delivery of all the required nutrients parenterally. It is a solution containing proteins, carbohydrates, fat, vitamins, and minerals. Because of the high osmolarity of the solution and the risk of phlebitis, it is usually given centrally rather than peripherally. Consequently, solutions delivered peripherally need to be diluted and may not meet the complete nutritional requirements of the patients.

When are TPN and PPN indicated?
TPN is indicated when patients need long-term nutritional support but are not able to receive enteral feedings (nonworking GI tract, shock, pancreatitis, bone marrow transplant, etc.). PPN is indicated in patients requiring short-term nutritional support (<10 days) to restrict protein breakdown.

- **Indications** of parenteral nutrition: indicated for patients who require nutritional support but cannot meet their needs through oral intake and for whom enteral feeding is contraindicated or not tolerated.
- **Contraindications**: heart failure, blood dyscrasia and altered fat metabolism.
- **Complications** of parenteral nutrition?
  Hyperglycemia, fatty liver, hypercapnia, acute respiratory distress syndrome, GI mucosal atrophy (predisposing the gut for bacterial translocation and septicemia). Catheter-related complications include infections and pneumothorax

Shock

Dr. sabah alheeti

Learning objective

✓ To understand what shock is, what causes it, and how it is best managed according to the cause.

Shock is characterized by inadequate perfusion of vital organs, principally the heart and brain.

Aetiology

Tissue perfusion requires an adequate blood pressure, which is dependent upon the systemic vascular resistance and cardiac output; the cardiac output is a function of the heart rate and the stroke volume. These may be expressed in mathematical terms: $CO = HR \cdot SV$

$BP = CO \cdot SVR$

where CO is cardiac output, SV is stroke volume, HR is heart rate, BP is arterial blood pressure and SVR is systemic vascular resistance.

Normal regulation of tissue perfusion

The autonomic nervous system is able to alter heart rate and peripheral vascular resistance in response to changes in blood pressure detected by the carotid sinus and aortic arch baroreceptors; changes in systemic vascular resistance may alter venous return by changing the amount of fluid circulating in the cutaneous and splanchnic vascular beds. Venous return determines stroke volume; increasing venous return causes an increase in stroke volume, the heart acting as a permissive pump (Starling ' s law: 1 the output depends on the degree of stretch of the heart muscle at the end of diastole). Volume regulation is achieved by the kidney, in particular by the regulation of sodium loss by the renin – angiotensin – aldosterone system and antidiuretic hormone (ADH) produced by the posterior pituitary; in addition, a fall in circulating volume prompts the sensation of thirst, stimulating increased fluid intake.

Abnormal regulation of tissue perfusion

Inadequate tissue perfusion (shock) may result from factors related to the pump (the heart) and factors relating to the systemic circulation. The causes of shock may be classified accordingly, as follows:

1 ***Cardiogenic shock.*** A primary failure of cardiac output in which the heart is unable to maintain adequate stroke volume in spite of satisfactory filling. Compensation involves an increase in heart rate and systemic vascular resistance, manifested clinically by a tachycardia, sweating (due to sympathetic nervous system outflow), pallor and coldness (due to cutaneous vasoconstriction). Causes include the following:

a massive myocardial infarction;

b pulmonary embolism;

c acute ventriculoseptal defect following myocardial infarction affecting the septum;
d mitral or aortic valve rupture;
e acute cardiac tamponade.

2 Hypovolemic (*Fluid loss*)*:* Reduction in circulating volume results in a reduction in stroke volume and cardiac output. Blood pressure is initially maintained as in cardiogenic shock, with increased sympathetic activity raising the peripheral vascular resistance leading to the clinical picture of a cold, clammy patient with a tachycardia. As volume losses increase, the blood pressure falls. In severe cases, the patient is confused or semiconscious. Causes include:
a haemorrhage, revealed or internal (e.g. ruptured aneurysm; bleeding into the bowel or around a closed fracture);
b burns, with massive loss of plasma and electrolytes;
c severe diarrhoea or vomiting, with fluid and electrolyte loss, particularly in colitis or pyloric stenosis;
d bowel obstruction, in which large amounts of fluid are sequestered into the gut, in addition to the losses due to vomiting;
e peritonitis, with large fluid losses into the abdomen as a consequence of infection or chemical irritation;
f gastrointestinal fistulae with fluid and electrolyte loss;
g urinary losses, e.g. the osmotic diuresis of diabetic ketoacidosis, or polyuria in resolving acute tubular necrosis.

3 *Reduction in systemic vascular resistance*. Reduction in systemic vascular resistance increases the size of the systemic vascular bed, producing a relative hypovolaemia, reduced diastolic filling, reduced stroke volume and thus a fall in blood pressure. Unlike the previous two causes, vasodilatation occurs as part of the pathogenesis, so the patient appears warm (' hot shock '), not cold and peripherally shut down. The heart compensates with an increase in output. The principal causes are:
a. anaphylaxis;
b. sepsis;
c. spinal shock. This It follows transection of the spinal cord (spinal shock), but may also occur after a high spinal anaesthetic. Sympathetic interruption occurs and this reduces the effective blood volume by widespread vasodilatation and bradycardia.

Septic shock

Shock may be produced as the result of severe infection from either Gram - positive or, more commonly, Gram - negative organisms. The latter are seen particularly after colonic, biliary and urological surgery, and with infected severe burns. The principal effect of endotoxins is to cause vasodilatation of the peripheral circulation together with increased capillary permeability.
The effects are partly direct and partly due to activation of normal tissue inflammatory responses such as the complement system and release of cytokines such as tumour necrosis factor (TNF).
Disseminated intravascular coagulation (DIC) results from activation of the clotting cascade and may lead to blockage of the arterial microcirculation by microemboli.

Fibrin and platelets are consumed excessively, with resultant spontaneous haemorrhages into the skin, the gastrointestinal tract, the lungs, mouth and nose.

Special causes of shock

1. Adrenocortical failure

Loss of the hormones produced by the cortex of the suprarenal gland may follow bilateral suprarenal haemorrhage, adrenalectomy, Addison ' s disease 2 or lack of corticosteroid replacement in patients who have been on long – term glucocorticoids. Failure of aldosterone secretion results in volume depletion and glucocorticoid deficiency, which impairs autonomic responses.
The ability to respond to minor stress is severely compromised and may provoke an Addisonian crisis characterized by bradycardia and postural hypotension, which is responsive to corticosteroid replacement. Adrenocortical failure should be considered and a bolus of hydrocortisone given in all patients with unexplained hypotension.

2.The vasovagal syndrome (faint)

The vasovagal syndrome is produced by severe pain or emotional disturbance. It is the result of reflex vasodilatation together with cardiac slowing owing to vagal activity. Hypotension is caused by a fall in cardiac output due to both bradycardia and reduced venous return; the latter the result of peripheral vasodilatation. Clinically, it is recognized by the presence of a bradycardia and responds to the simple measure of laying the patient flat with elevation of the legs.

Sequelae of shock

A continued low blood pressure produces a series of irreversible changes, so that the patient may die in spite of treatment. The lack of oxygen affects all the vital organs.
 Cerebral hypoperfusion results in confusion or coma.
 Renal hypoperfusion results in reduced glomerular filtration, with oliguria or anuria. As renal ischaemia progresses, tubular necrosis may occur, and profound ischaemia may lead to cortical necrosis.
 The heart may fail owing to inadequate coronary perfusion.
 Pulmonary capillaries may reflect the changes in the systemic circulation with transudation of fluid resulting in pulmonary oedema, hampering oxygen transfer and causing further arterial hypoxaemia and thus tissue hypoxia.
Pulmonary capillary function may also be impaired following multiple blood transfusions and contusions resulting from chest trauma, a condition known as acute lung injury (previously termed ' shock lung ').
 DIC , precipitated by sepsis, may be further aggravated by hypothermia unless active re - warming is undertaken.

Comparison of different types of shock

Hypovolemic	Hypotension, tachycardia Weak thready pulse Cool, pale, moist skin U/O decreased	Decreased CO **Increased SVR** **Decreased CVP**
Cardiogenic	Hypotension, tachycardia Weak thready pulse Cool, pale, moist skin U/O < 30 ml/hr Crackles, tachypnea	Decreased CO **Increased** SVR **Increased CVP**
Neurogenic	Hypotension, BRADYCARDIA **WARM DRY SKIN**	Decreased CO **Venous & arterial vasodilation, loss sympathetic tone**
Anaphylactic	Hypotension, tachycardia Cough, dyspnea Pruritus, urticaria Restlessness, decreased LOC	Decreased CO **Decreased** SVR
Septic	Hypotension, Tachycardia Full bounding pulse, tachypnea **Pink, warm, flushed skin** Decreased U/O, fever	**Decreased** CO, **Decreased** SVR

Principles in the management of patients in shock

Immediate measures

The immediate treatment of patients in shock varies according to cause. Two causes merit mention for immediate treatment: bleeding and anaphylaxis.

Bleeding

Direct pressure should be applied to a bleeding wound. Immediate surgical exploration is indicated where continued bleeding is likely, such as in peptic ulcer haemorrhage, ruptured spleen, ruptured aortic aneurysm or ruptured ectopic pregnancy. In these cases, resuscitation cannot overcome the losses until the rate of blood loss is curtailed.

Anaphylaxis

In surgical practice, this may arise as an allergic reaction to an antibiotic or radiological contrast medium. In addition to hypotension (due to vasodilatation), bronchospasm and laryngeal oedema may be present and warrant immediate therapy. The immediate treatment for anaphylaxis is the administration of adrenaline (epinephrine; 0.5 mL of 1:1000 concentration)

intramuscularly or subcutaneously, repeated every 10 – 30 minutes as required. Subsequently, hydrocortisone and antihistamine agents may be given (e.g. chlorphenamine).
For milder reactions, aliquots of 1 mL of 1:10 000 adrenaline are given and titrated to effect.

Monitoring and subsequent management

The severely shocked patient should be admitted to an intensive care ward where continuous supervision by specially trained nursing staff is available. As well as careful clinical surveillance, the following need to be monitored:

- Core temperature, pulse, respiration rate and blood pressure.
- Hourly urine output (via a urinary catheter).
- Central venous pressure.
- Pulse oximetry. Oxygen is administered to ensure adequate oxygenation. Mechanical ventilation may be required.
- Electrocardiogram (ECG).
- Serum electrolytes, haemoglobin and white blood cell count.
- Arterial blood gases (*P* o2 , *P* co2 , [H +]).
- The cardiac output, and left atrial and pulmonary arterial pressures using a Swan – Ganz catheter.

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Pharmacological agents

The shocked patient may require significant pharmacological support. The principal drugs used are catecholamines or their derivatives, in addition to drugs to treat specific causes such as antimicrobial therapy for septicaemia. Patients in cardiogenic shock benefit from positive inotropic agents, whereas patients with low systemic vascular resistance due to sepsis require agents to increase vascular resistance. The drugs used in this context are sympathomimetics, with differing degrees of α (peripheral vasoconstriction), β 1 (inotropic and chronotropic) and β 2 (peripheral vasodilatation) effects. Examples of such drugs include the following.

Dopamine

Dopamine has three separate actions according to dose:
1-At *low doses* (2 μ g/kg/min) dopaminergic actions dominate, causing increased renal perfusion. This is the commonest indication for the use of dopamine.
2- At *moderate doses* (5 μ g/kg/min), β1 effects predominate with positive inotropic activity (increasing myocardial contractility and rate).
3- At *higher doses* (over 5 μ g/kg/min), α effects predominate with vasoconstriction.

Dobutamine

Dobutamine has predominantly β 1 actions, increasing myocardial contractility and rate, thus increasing cardiac output. It is used principally in cardiogenic shock.

Noradrenaline (norepinephrine)

Noradrenaline has predominantly α effects, but with modest β activity. It is used to increase systemic vascular resistance through its vasoconstrictor α effects.

Adrenaline (epinephrine)

Adrenaline has strong α and β actions, and may be used to increase peripheral resistance while also increasing cardiac output. The powerful vasoconstrictor actions of both adrenaline and noradrenaline may result in ischaemia and infarction of peripheral tissues, most commonly fingers, toes and the tips of the nose and ears.

Surgical infection

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Surgical infections : infections that require operative treatment or result from operative treatment.

Infections that require operative treatment include:

- necrotizing soft tissue infections
- body cavity infections such as peritonitis, suppurative pericarditis and empyema.
- confined tissue, organ, or joint infections such as abscess and septic arthritis
- prosthetic device–associated infections e.g. infected plate used in fixation of fracture

infections that result from operative treatments include:

- wound infections,
- postoperative abscesses,
- postoperative body cavity infections such as postoperative peritonitis.
- prosthetic device– related infections,
- hospital-acquired infections such as pneumonia.

Determinants of Infection

The development of surgical infection depends on several factors:

(1) microbial pathogenicity and number,

(2) host defenses,

(3) the local environment: e.g a traumatic wound has greater likelihood if the trauma has resulted in devitalization of tissue or if foreign bodies have been deposited in the wound.

(4) surgical technique: the surgeon can reduce the likelihood of postoperative infections by handling tissues gently; removing devitalized tissues, blood and other substances that promote growth of microbes; and using drains appropriately.

Types of surgical infections

Soft tissue infections include:

1- Cellulitis and lymphangitis: Cellulitis is a non-suppurative, invasive infection of tissues, which is usually related to the point of injury.There is poor localisation in addition to the cardinal signs of spreading inflammation (redness, heatness, swelling, pain and loss of function). Systemic signs (the old-fashioned term is toxaemia) are common, with chills, fever and rigors. These events follow the release of toxins into the circulation.

Lymphangitis is part of a similar process and presents as painful red streaks in affected lymphatics draining the source of infection. Lymphangitis is often accompanied by painful lymph node groups (lymphadenitis) in the related drainage area. Blood cultures are often negative.

Treatment: antibiotics alone but Surgical incision and drainage are indicated if no improvement is seen in 2 to 3 days, or if evidence of purulent collection is identified.

2- Soft tissue abscess:

An abscess is a pocket of tissue containing necrotic tissue, bacterial colonies, and dead white cells.

The area of infection may or may not be fluctuant. The patient is often febrile at this stage.

Treatment: incision and drainage (I&D) and leave the cavity to heal by secondary intention. Antibiotics should be used if the abscess cavity is closed after drainage.

Table 28-3. A Comparison of Cellulitis and Abscess

	CELLULITIS	ABSCESS
Duration	Acute	Chronic
Pain	Severe and generalized	Localized
Size	Large	Small
Localization	Diffuse borders	Well circumscribed
Palpation	Doughy to indurated	Fluctuant
Presence of pus	No	Yes
Degree of seriousness	Greater	Less
Bacteria	Aerobic	Anaerobic

3- Necrotizing Soft tissue infections (gas gangrene, necrotizing fasciitis)

They can be defined as infections of any of the layers within the soft tissue compartment (dermis, subcutaneous tissue, superficial fascia, deep fascia, or muscle) that are associated with necrotizing changes. NSTIs are typically not associated with abscesses, although they can originate from an untreated or inadequately drained abscess. Debridement of the necrotic tissue should be undertaken as soon as possible together with other principles of treatment for any kind of surgical infection: source control, antimicrobial therapy, support.

Antibiotics in surgical infections

A. **Indications :** is used only as adjunct in treating surgical infection; operative treatment is more important. The antibiotics is used either for
   I. *Treatment of infection (Empiric treatment, Definitive treatment).*
   II. *Prophylactic antibiotics*

B. ***Principles of antibiotic treatment***
   I. *Identify most likely causative organism (bacteria, fungus or virus)*

II. *Use appropriate antibiotic agents*
III. *Initially, start antibiotic treatment on presenting identifications and clinical judgment.*
IV. *Assessment of renal and hepatic functions*
V. *Presence of hypersensitivity to drugs.*
VI. *Prior to treatment specimens of blood, urine should be collected.*
VII. *Pus should be drained*
VIII. *Necrotic tissue and foreign bodies should be removed.*
IX. *Once started AB. , should never be changed unless features of no responding by clinical examinations or culture results show different pathogens.*
X. *Route of giving antibiotics*

C. Complications of antibiotic treatment

I. *Development of resistance.*
II. *Hypersensitivity.*
III. *Side and irritation effects.*
IV. *Opportunistic infections (disturbe normal flora).*
V. *Toxic effects*

D. Indications of combined antibiotic treatment

I. *Treatment of mixed infections.*
II. *To delay development of bacterial resistance.*
III. *Initial treatment of serious infections.*
IV. *To obtain potentiation or synergistic actions*

E. Causes of ineffective antibiotic treatment

I. *Wrong route of administration.*
II. *Impaired host defence mechanism.*
III. *Abscess not adequately drained.*
IV. *Presence of foreign body.*
V. *Delay in initiation of treatment.*
VI. *Improper dose given*

F. What are the indications for prophylactic antibiotics?

I. *when bacterial contamination of the wound is high(clean-contaminated, contaminated and dirty wounds)*
II. *for patients having clean operations in which a prosthetic device is placed*

G. Principles of prophylactic antibiotics:

I. *Prophylactic antibiotic therapy should be directed against the bacteria likely to contaminate the wound.*

II. *The antibiotics usually should be given intravenously 30– 60 min before operation . it should not be continued beyond the day of operation.*
III. *The value of antibiotic prophylaxis is low in non-prosthetic clean surgery, while they are effective in reducing the risk of infection in clean-contaminated and contaminated operations.*
IV. *Cephalosporins are the most commonly used antibiotics for prophylaxis because of their broad antibacterial spectrum.*
V. *In long operations or when there is excessive blood loss, or when unexpected contamination occurs, antibiotics may be repeated at 4-hourly intervals during the surgery, because tissue antibiotic levels often fall faster than serum levels.*
VI. *Patients with known valvular disease of the heart (or with any implanted vascular or orthopaedic prosthesis) should have prophylactic antibiotics during dental, urological or open viscus surgery, to prevent bacterial colonisation of the valve or prosthesis during the transient bacteraemia which can occur during such surgery*

Opportunistic infection

An opportunistic infection is an infection caused by pathogens (bacteria, viruses, fungi, or protozoa) that take advantage of an opportunity not normally available, such as a host with a **weakened immune system**, an **altered microbiota** (such as a disrupted gut microbiota), or **breached integumentary barriers** (due to injury or medical procedure like cannula, folley catheter). Many of these pathogens do not cause disease in a healthy host that has a normal immune system.

HOSPITAL-ACQUIRED (NOSOCOMIAL) INFECTIONS

The infection that is acquired in operative theatre and/or wards . The most important and frequent mode of transmission of nosocomial infections is by direct contact. Others routes of transmission are Droplet transmission, airborne, vehicle transmission(food, mediactions..) and vector borne (such as mosquitoes, flies). The most common nosocomial infections are:

I. urinary tract infections are most common, followed by
II. wound infections
III. lower respiratory infections such as ventilator associated pneumonia
IV. bacteremia, and cutaneous infections

Surgical wound infections

The wounds have been classified into 4 categories according to the theoretical number of bacteria that contaminate wounds:

Wound Class	Definition	Examples of Typical Procedures	Wound Infection Rate (%)	Usual Organisms
Clean	Nontraumatic, elective surgery; no entry of GI, biliary, tracheobronchial, respiratory, or GU tracts	Wide local excision of breast mass	2	*Staphylococcus aureus*
Clean-contaminated	Respiratory, genitourinary, GI tract entered but minimal contamination	Gastrectomy, hysterectomy	<10	Related to the viscus entered
Contaminated	Open, fresh, traumatic wounds; uncontrolled spillage from an unprepared hollow viscus; minor break in sterile technique	Ruptured appendix; resection of unprepared bowel	20	Depends on underlying disease
Dirty	Open, traumatic, dirty wounds; traumatic perforated viscus; pus in the operative field	Intestinal fistula resection	28–70	Depends on underlying disease

Wound Healing

The healing wound is cellular and biochemical responses directed toward restoring tissue integrity and functional capacity following injury.
Injured organisms survive only if they can repair themselves quickly and effectively. The healing response depends primarily on the type of tissue involved and the nature of the tissue disruption.
When restitution occurs by means of tissue that is structurally and functionally indistinguishable from native tissue, ***regeneration*** has taken place. However, if tissue integrity is reestablished primarily through the formation of fibrotic scar tissue tissue, Then ***repair*** has occurred.
With the exception of bone and liver, tissue disruption invariably results in repair rather than regeneration

Wound healing involves three overlapping phases :
1- Inflammation,
2- Proliferation
3- Remodelling.
This produce scar which represent trace of healing wound.

Inflammatory phase

1. Begins at the time of injury; lasts 2 to 3 days.
2. Begins with vasoconstriction to achieve hemostasis (epinephrine and thromboxane).
3. Platelet plug forms and clotting cascade is activated, resulting in fibrin deposition.
4. Platelets release platelet-derived growth factor (PDGF) and transforming growth factor β (TGF-β) from their alpha granules, attracting inflammatory cells, particularly macrophages.
5. After hemostasis is achieved, vasodilation occurs and vascular permeability increases (due to histamine, platelet-activating factor, bradykinin, prostaglandin I_2, prostaglandin E_2, and nitric oxide), aiding the infiltration of inflammatory cells into the wound.
6. Neutrophils peak at 24 hours and help with débridement.
7. Monocytes enter the wound, becoming macrophages, and peak within 2 to 3 days.
8. Limited numbers of lymphocytes arrive later, but their significance is unknown.

Proliferative phase

1. Begins around day 3, as fibroblasts arrive; lasts through week 3.
2. Fibroblasts: Attracted and activated by PDGF and TGF-β; arrive day 3, reach peak numbers by day 7.
3. Collagen synthesis (mainly type III), angiogenesis, and epithelialization occur.
4. Total collagen content increases for 3 weeks, until collagen production and breakdown become equal and the remodeling phase begins.

Remodeling phase

1. Increased collagen production and breakdown continue for 6 months to 1 year.
2. Type I collagen replaces type III until it reaches a 4:1 ratio of type I to type III (that of normal skin and mature scar tissue).

3. Wound strength increases as collagen reorganizes along lines of tension and is cross-linked.
4. Vascularity decreases.
5. Fibroblast and myofibroblasts cause wound contraction during the remodeling phase.
6- Wounds have 50% of their final strength at 6 weeks, but never reaches normal (only 80% normal).

Fetal wound healing

A. Skin (but not all fetal tissue) heals by regeneration without scarring. This is limited to the first two trimesters.
B. Many aspects of fetal tissue and the fetal environment may contribute to scarless healing.
1. The fetal environment (amniotic fluid) is sterile.
2. Amniotic fluid contains growth factors and extracellular matrix molecules.
3. The inflammatory phase is minimal, and macrophages may or may not be the main organizing cells in the healing process in the fetus.
4. The growth factor and cytokine milieu is different in the fetus, although the significance of any particular difference is unclear

TYPES OF WOUND HEALING

A. Primary Healing
Wounds are closed by reapproximation using suture or by some other mechanical means within hours of their creation. This is indicated in newly created wounds (<24hrs), clean wound and when there is no tissue loss.
B. Delayed primary closure (tertiary healing)
1. Wound remains open for a few days before surgical closure.
2. Decreases the risk of infection in contaminated wounds.
C. Secondary closure
1. Wound closes over time by contraction and epithelialization.
2. Appropriate for infected or contaminated wounds.
3. Allows drainage of fluid.
4. Allows debridement with dressing changes.
5. Prolonged inflammatory phase, leading to increased scarring and wound contracture.

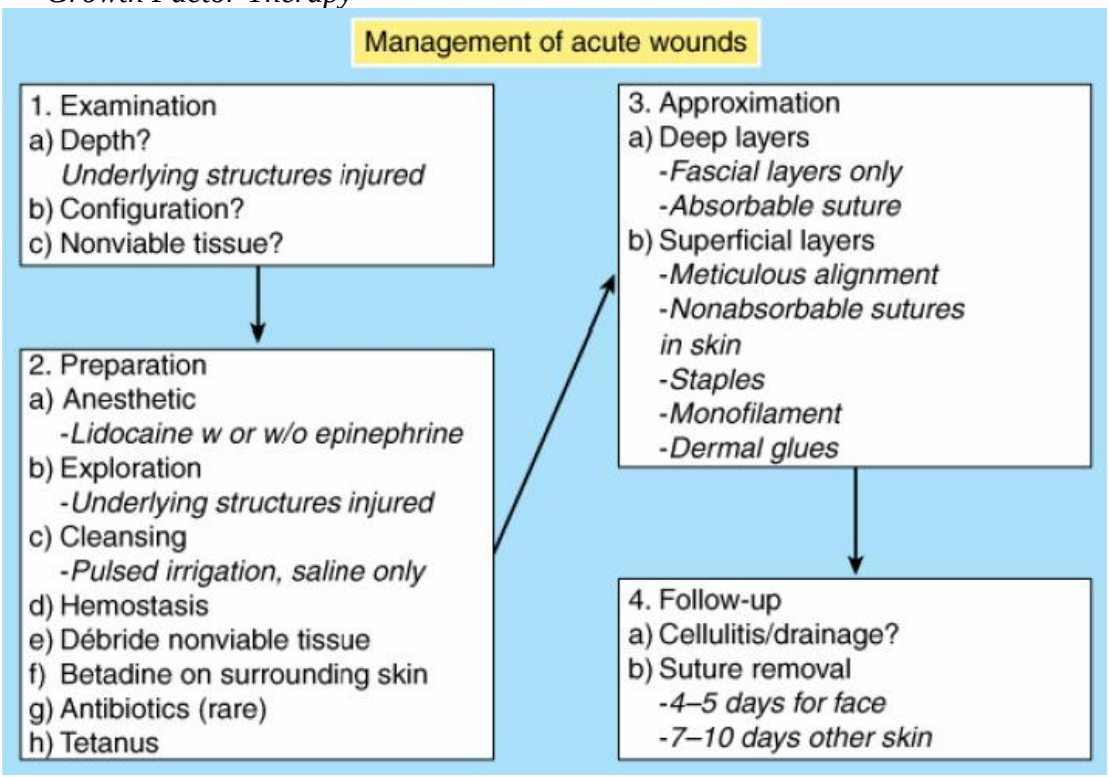
Factors Contributing to Impaired Wound Healing

I. Local factors
A-Mechanical injury
B- Infection
C- Edema
D- Ischemia//necrotic tissue
E- Topical agents
F- Ionizing radiation
G- Low oxygen tension
H- Foreign bodies
II. Systemic factors
A- Age
B- Nutrition

C- Trauma
D- Metabolic diseases
E- Immunosuppression
F- Connective tissue disorders
G- Smoking

TREATMENT OF WOUNDS:

- *Local Care*
- *Antibiotics*
- *Dressings*
- *Skin Replacements*
- *Growth Factor Therapy*



Excessive Wound Healing

I. Keloids

A. Excessive scar formation: Defined by scar tissue that extends beyond the boundaries of the incision or wound.
B. Etiology
1. Not completely understood, but growth factors certainly play a role.
2. Keloids contain elevated levels of TGF-β.
C. Demographics and natural history
1. More common in patients of African ancestry.
2. Tendency to form keloids is often inherited in an autosomal dominant pattern.
3. Common in ear lobes and areas of tension.
4. Keloids may develop months to a year after injury, and do not resolve spontaneously.
D. Histology

1. Excess collagen, and increased vascularity compared with normal scar tissue.
2. Collagen production is many times that seen in normal scar tissue, and there is a higher proportion of type III collagen.
E. Treatment
1. Excision alone is rarely successful.
2. Corticosteroid injection may cause some reduction in keloid size.
3. Excision followed by corticosteroid injection locally is more successful.
4. Excision should be followed by radiation therapy for severe cases.
5. Recurrence is common.

II. Hypertrophic scars

A. Excessive scar formation: Defined by scar tissue that does not extend beyond the boundaries of the incision or wound.
B. Etiology
1. Prolonged or increased inflammatory phase of healing.
2. Increased wound tension.
C. Demographics and natural history
1. More common in patients of African ancestry.
2. Less genetic component than keloids.
3. Tendency decreases with age, as the inflammatory phase of healing decreases.
4. More common in areas of tension, such as the presternal area.
5. Develop within weeks of wounding (during the inflammatory phase), and there is usually some degree of improvement with time.
D. Histology
1. Increased collagen with collagen nodules, hypervascularity.
2. Collagen production is increased compared with normal scar tissue, but less than in keloids.
E. Treatment
1. Corticosteroid injection, silicone sheeting, and pressure are often successful in reducing the degree of scar hypertrophy. Multiple treatments with corticosteroids may be required, and silicone sheeting and pressure garments must be applied for at least 6 months before improvement is seen.
2. Surgical excision and reclosure may be successful if nonsurgical modalities are not working, and if the wound can be closed without tension.